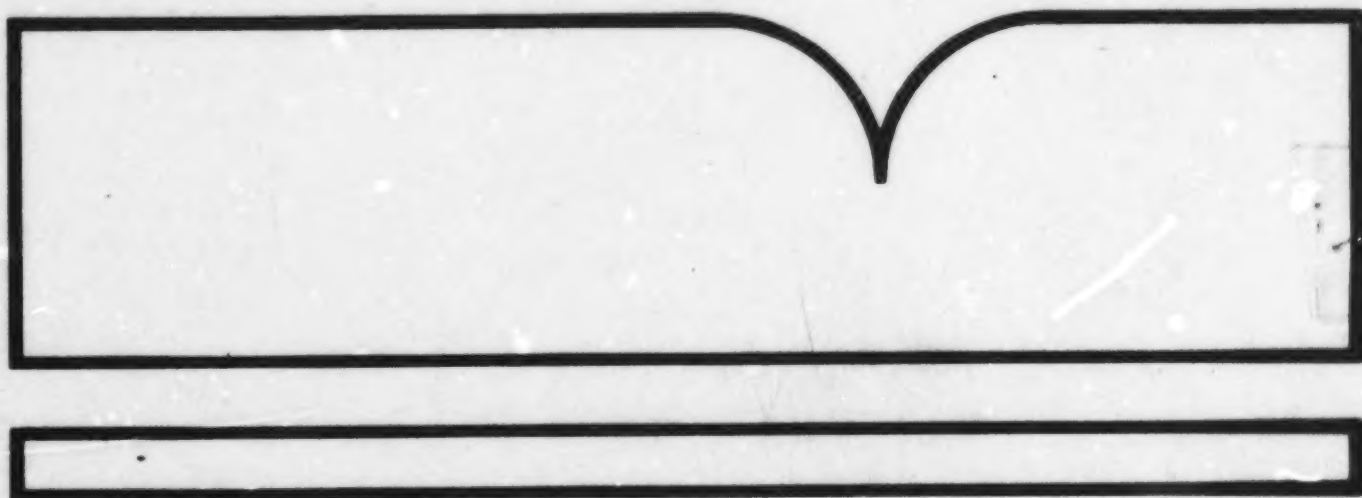


Biochemical Effects of Inhalation of Sulfuric
Acid Mist by Human Subjects While at Rest

(U.S.) Health Effects Research Lab.
Research Triangle Park, NC

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EPA-600/1-79-042
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**BIOCHEMICAL EFFECTS OF INHALATION OF SULFURIC ACID MIST
BY HUMAN SUBJECTS WHILE AT REST**

by

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Wendy Blomquist
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FOREWORD

The many benefits of our modern, developing, industrial society are accompanied by certain hazards. Careful assessment of the relative risk of existing and new man-made environmental hazards is necessary for the establishment of sound regulatory policy. These regulations serve to enhance the quality of our environment in order to promote the public health and welfare and the productive capacity of our Nation's population.

The Health Effects Research Laboratory, Research Triangle Park, conducts a coordinated environmental health research program in toxicology, epidemiology, and clinical studies using human volunteer subjects. These studies address problems in air pollution, non-ionizing radiation, environmental carcinogenesis and the toxicology of pesticides as well as other chemical pollutants. The Laboratory participates in the development and revision of air quality criteria documents on pollutants for which national ambient air quality standards exist or are proposed, provides the data for registration of new pesticides or proposed suspension of those already in use, conducts research on hazardous and toxic materials, and is primarily responsible for providing the health basis for non-ionizing radiation standards. Direct support to the regulatory function of the Agency is provided in the form of expert testimony and preparation of affidavits as well as expert advice to the Administrator to assure the adequacy of health care and surveillance of persons having suffered imminent and substantial endangerment of their health.

This report addresses itself to the identity and the measurement of possible health effects of sulfuric acid (H_2SO_4) aerosol in humans.

F. G. Hueter, Ph.D.
Director
Health Effects Research Laboratory (MD-51)

ABSTRACT

A total of 20 human subjects were exposed to $100 \mu\text{g}/\text{m}^3$ ($0.033 \mu\text{M}$) sulfuric acid aerosol ($0.5 \mu\text{m}$ mean mass diameter) for four hours per day for two consecutive days. A total of 17 human subjects were exposed to four hours of ambient air on both exposure days. The subjects were at rest during both exposures.

Seven biochemical blood parameters were measured pre and post exposure and 20 hours after the second exposure: serum glutathione, red blood cell glutathione reductase, red blood cell glucose-6-phosphate dehydrogenase, lysozyme, serum glutamic oxaloacetic acid transaminase, serum vitamin E and 2,3-diphosphoglycerate.

The results indicate no significant response in any of the seven biochemical blood parameters measured.

This report covers the period of January 15, 1979 to March 23, 1979 and work was completed as of May 1, 1979.

INTRODUCTION

With the increase of automobile catalytic converters and the use of high sulfur content coal as an energy source, the peak levels of sulfuric acid (H_2SO_4) could foreseeably rise by as much as three to four times the present level. Thus, it becomes necessary to identify and measure the possible health effects of H_2SO_4 aerosol in humans.

Evidence for any aggravation and/or causation of changes in human health induced by H_2SO_4 aerosol has been sparse. The majority of the biochemical parameters measured after H_2SO_4 aerosol exposure have been in animal models. Early animal H_2SO_4 exposure studies indicated there is a hierarchy of toxicity (guinea pigs > mice > rats > rabbits).¹ In addition it was reported that the toxicity within a species is age dependent.² Lewis et al.³ measured hematological changes and pulmonary function in female beagles exposed to 13.4 mg/m^3 sulfur dioxide (SO_2) and 0.9 mg/m^3 H_2SO_4 alone and in combination. They concluded that (1) chronic exposure to relatively low levels of H_2SO_4 can produce serious pulmonary impairment; (2) lungs previously exposed to a toxic agent, NO_2 in their study, are less likely to show altered lung physiology upon subsequent exposure to low levels of airborne pollutants; and (3) no significant changes in hematology occurred as a result of the exposure.

Alorie et al.⁴ reported that long term exposures of cynomolgus monkeys and guinea pigs to H_2SO_4 mist at concentrations varying from 0.38 to 4.79 mg/m^3 produced no changes in hematological parameters while the routine biochemical screening studies showed considerable variation but no discernible trends.

Human exposure studies reported in the literature have concentrated on pulmonary function effects. Amdur et al.¹⁵ reported respiration becomes rapid and shallow in normal men exposed to 350 to 5000 $\mu\text{g}/\text{m}^3$ H_2SO_4 . Increased respiration rate results in decreased retention of inhaled particles. It would then appear possible that the more rapid, shallower breathing that was observed is a reflex protective mechanism to prevent retention of the acid mist.

Sim and Pattle¹⁶ studied the response of healthy males aged 18 to 46 to H_2SO_4 mist. Subjects were either exposed to H_2SO_4 mist for 10 minutes by mask or exposed to H_2SO_4 mist for 60 minutes in an exposure chamber. They measured the irritant effect of H_2SO_4 mist at a relative humidity of 62% or 90%. They found H_2SO_4 to be more irritating under conditions of high humidity. Coughing was continuous throughout the exposure period. No airway resistance measurements could be made during the first 10 minutes. When the coughing diminished enough to permit measurement, the increases ranged from 43% to 150% above normal.

This study was undertaken to evaluate the effects of sulfuric acid aerosol on seven human biochemical blood parameters.

METHOD

Subjects

A total of 37 healthy, non-smoking, Caucasian male university students characterized by an average height of 180.6 cm (a standard deviation of 5.1 cm, a range of 168.0 to 194.0 cm), an average weight of 73.0 kg (a standard deviation of 8.0 kg, range 56.5 to 89.5 kg), and an average age of 22.4 years (a standard deviation of 3.2 years, a range of 18.4 to 29.7 years), were used in this study. A total of 17 subjects were exposed to air (group 1, controls) and a total of 20 subjects were exposed to $100 \mu\text{g}/\text{m}^3 \text{H}_2\text{SO}_4$ aerosol (group 2, experimental).

Procedure

Each subject was exposed at rest for 4 hours on two consecutive days. On each day, blood was drawn immediately preceding the four hour exposure and immediately following the exposure. A 20 hour post exposure blood sample was drawn the morning after the second exposure day. Subjects were tested in groups of three.

Experimental Design

The control group of subjects was exposed to ambient air on the first and second days of exposure. The experimental group was exposed to two consecutive days of $100 \mu\text{g}/\text{m}^3 \text{H}_2\text{SO}_4$ aerosol ($0.5 \mu\text{m}$ mean mass diameter, MMD). The exposure began and ended the same time each day. Data were collected in a counterbalanced fashion. During Week 1, a group of three acid exposure subjects was tested on Monday and Tuesday and a group of three air exposure subjects were tested on Wednesday and Thursday. The following week the order was reversed. This alternation

scheme was continued throughout the experiment. This scheme helped control any day-of-week effects and time-of-year effects.

The chamber atmosphere was maintained at a temperature of 20°C, a relative humidity of 40% and an air flow of 227 m³/min.

A total of seven serum and red blood cell biochemical measurements were chosen as dependent variables: serum glutathione (GSH), red blood cell glutathione reductase, red blood cell glucose-6-phosphate dehydrogenase (G6PDH), lysozyme, serum glutamic oxaloacetic acid transaminase (SGOT), serum vitamin E, and 2,3-diphosphoglycerate (2,3-DPG). Measurements were taken on all seven variables preceding and following both exposure days and 20 hours after the second exposure day.

Multivariate analysis of variance (MANOVA) was used to analyze the results. The seven blood parameters were each measured five times to give 35 dependent variables. Three a priori contrasts were chosen to assess (1) the initial difference between the air and acid groups, (2) a time effect for air only and (3) an acid effect, i.e. the group by time interaction.

Blood Analysis

GSH in serum was measured by the method of Patterson and Lazarow.^{5,6} Red blood cell glutathione reductase was assayed by the method of Nichoalds⁷ modified such that the enzyme was preincubated at 0°C for 30 minutes in the presence of 60 µm FAD and the reaction was initiated by the simultaneous addition of 2 mM oxidized glutathione (GSSG) and 0.24 mM NADPH. Red blood cell G6PDH was assayed by the procedure of Catalano,⁸ lysozyme by the method of Shugar,⁹ serum vitamin E by the method of Chaney,¹⁰

2,3-DPG in erythrocytes by the Nygaard and Rorth¹¹ method. SGOT was analyzed on the Centrifichem 400 autoanalyzer by a modified Karmen technique.^{12,13}

Materials

A Centrifichem Model 400 centrifugal analyzer and Centrificham autopipetter (Union Carbide Corporation, Clinical Diagnostics Division, Rye, New York 10580) were used for assaying SGOT and G6PDH. Manual assays for glutathione reductase, GSH, and 2,3-DPG were run on a Cary 118 spectrophotometer. The lysozyme assay was run on a Varian 635 spectrophotometer.

SGOT reagent kits were purchased from Union Carbide Corporation, Clinical Diagnostics Division, Rye, New York 10580. 2,3-DPG reagent kits were purchased from Calbiochem, La Jolla, California 92037. Lysozyme reagent kits were purchased from Worthington Diagnostics, Freebold, New Jersey 07728.

D, L- α -tocopherol, GSSH (free acid, grade III), FAD (disodium salt, grade III), NADPH (tetrasodium salt, type 1), NADP⁺ (monosodium salt, Sigma Grade), 6-phosphogluconic dehydrogenase, Type V from yeast, 50 U/ml, D-glucose-6-phosphate monosodium salt, alloxan monohydrate and GSH (reduced form, 98-100% purity) were purchased from Sigma Chemical Company, St. Louis, Missouri 63178.

RESULTS

Table 1 provides summary statistics for all seven dependent variables for the initial blood draw. Data from all 37 subjects has been combined since none of the subjects had been exposed to any treatment at the time of this initial blood draw. The means, standard deviations, ranges, and skewnesses are reasonable for the purposes of the MANOVA.

Table 2 summarizes the MANOVA for the seven dependent variables. All tests presented are multivariate tests, i.e., all seven variables are treated simultaneously. The three contrasts computed require explanation. The first contrast is labeled group difference at pre-1. This tests the hypothesis of no difference on any of the variables for the treatment and control groups (H_2SO_4 group and air group). Since this test is not significant ($p = .20$), the hypothesis that the two groups began the experiment with the same values for the variables measured may not be rejected.

The second contrast tested evaluates the presence of any difference across time for the air exposure group by itself. This is, therefore, the simple effect of time within air. Since the multivariate test is not significant ($p = .51$), the hypothesis that the mean levels are equal for all time points cannot be rejected.

The third contrast tests for any effect of H_2SO_4 exposure. This is the group by time interaction. This interaction was determined insignificant ($p = .47$), thus indicating no acid effect on any variable.

Table 3 provides mean response on all seven dependent variables for each group at each time point. These same means are presented graphically in Figure 1 through 7. These results indicate only small variations across time and groups. The standard deviations in Table 1 are representative of the standard deviations associated with the other time points.

The results indicate no response observed in any of the seven biochemical blood parameters measured as a result of the two 4-hour exposure to $100 \mu\text{g}/\text{m}^3$ ($0.033 \mu\text{M}$) H_2SO_4 aerosol (0.5 MMD) on two consecutive days. Similar results were obtained in a study in which human subjects were exposed to one 4-hour exposure to $100 \mu\text{g}/\text{m}^3$ H_2SO_4 aerosol.¹⁴

There are two possible ways that nonsignificant results can arise: (1) if there is great variability due to lack of precision, then all but very large effects will be masked; (2) on the other hand, with good precision, a nonsignificant result is indicated by very small differences among groups. In the latter case, if any effects do exist, they are very small. In the former case, one may only report that no effects were detectable. The authors feel that these data conform to the second pattern.

The results reported are consistent with the available animal studies. Even long term continuous exposure of monkeys to sulfuric acid mist at concentrations varying from 380 to $4790 \mu\text{g}/\text{m}^3$ (submicronic particles to 4μ MMD) and guinea pigs to concentrations at 80 and $100 \mu\text{g}/\text{m}^3$ with MMD of 0.8μ and 2.8μ respectively resulted in no trend

indicating possible effects from sulfuric acid on hematologic and serum biochemical parameters.⁴

Earlier human exposure studies reported pulmonary function effects only. The authors are not aware of any studies on the response of human blood parameters upon exposure to H_2SO_4 mist.

In reality, the effects of H_2SO_4 mist may not go beyond the upper respiratory tract. As has been reported earlier,¹⁴ the ammonia released by the respiratory system may well eliminate any effect of inhaled sulfuric acid aerosol through partial or complete neutralization. The findings of Pattle et al.¹⁷ support this hypothesis. While measuring the eight-hour lethal concentration 50% probable for guinea pigs exposed to H_2SO_4 , it was found that ammonium carbonate present in the chamber in an amount sufficient to provide an excess of ammonia gave protection from levels of H_2SO_4 which in the absence of ammonia, would have caused 50% mortality.

In conclusion, no significant effect of H_2SO_4 mist was found for any of the blood parameters measured. This finding is consistent with previous animal studies on biochemical blood parameters. Earlier studies involving humans have indicated pulmonary function effects, however, no previous results appear to have been published on human biochemical blood parameters. It is possible that, at the level of H_2SO_4 studied, the body's defense mechanisms limit any effects to the upper airways.

BIBLIOGRAPHY

1. Treon, J. F., F. R. Dutra, J. Chappel, Arch. Indust. Hyg. Occup. Med., 2, 716 (1950).
2. Amdur, M. O., R. Z. Schellz, P. Drinker, Arch. Indust. Hyg. Occup. Med., 5, 318 (1952).
3. Lewis, T. R., W. J. Moorman, W. F. Ludmann, K. I. Campbell, Arch. Environ. Health, 26, 16 (1973).
4. Alarie, Y., W. M. Busey, A. A. Krumm, C. E. Ulrich, Arch. Environ. Health, 27, 16 (1973).
5. Patterson, J., W. and A. Lazarow. Methods of Biochem. Anal., G. Guich, ed., N.Y. Interscience, 2, 259 (1955).
6. Patterson, J. W., A. Lazarow, S. Levey, JBC, 177, 197 (1949).
7. Nichoalds, G. E., Clin. Chem. 20, 624 (1974).
8. Catalano, E. W., G. T. Johnson, H. M. Solomon, Clin. Chem., 21, 134 (1975).
9. Shugar, D., Biochem. Biophys. Acta, 8, 302 (1952).
10. Chaney, S. Q., P. J. DeWitt, W. Blomquist, K. Muller, R. M. Bruce, G. M. Goldstein. Manuscript in preparation.
11. Nygaard, S. F. and M. Rorth, Scand. J. Clin. Lab. Invest., 24, 399 (1969).
12. Karmen, A., J. Clin. Invest., 34, 131 (1955).
13. Henry, R. J., N. Chiamori, O. J. Golub, S. Berkman, Am. J. Clin. Path., 34, 381 (1960).
14. Chaney, S. Q., W. Blomquist, K. Muller, G. Goldstein, U. S. Environmental Protection Agency. Report in preparation.

15. Amdur, M. O., L. Silverman, P. Drinker, Indust. Hyg. Occup. Med., 6, 305 (1952).
16. Simm, V. M., R. E. Pattle, JAMA, 165, 1908 (1957).
17. Pattle, R. E., F. Burgess, H. Cullumbine, J. Path. Bact., 72, 219 (1956).

TABLE 1
Summary of Statistics for All Seven Dependent Variables for Pre Day 1
(n = 37)

<u>Variable</u>	<u>Mean</u>	<u>Std. Dev.</u>	<u>Min.</u>	<u>Max.</u>	<u>Skewness</u>
GSH mg/100 ml	38.45	4.80	29.46	47.97	0.10
GSH Reductase μmole/gm Hgb/min	7.45	3.19	1.77	12.95	-0.28
RBC G6PDH U/gm Hgb	2.04	0.26	1.54	2.77	0.47
Lysozyme μg/ml	9.67	2.04	5.41	14.78	0.64
SGOT IU/l	11.7	2.5	6.5	17.5	0.51
Vitamin E μg/ml	5.22	1.51	2.70	9.78	0.69
2,3-DPG μmole/ml RBC	4.61	0.92	3.04	7.81	1.14

TABLE 2
MANOVA Summary for All Seven Dependent Variables

<u>Source</u>	<u>Likelihood Ratio</u>	<u>Λ F*</u>	<u>Num df</u>	<u>Den df</u>	<u>p</u>
Group Diff at Pre-1	.784	1.51	6	33	.20
Air Group Time Effect	.384	1.00	24	15	.51
Acid Effect (Gp x Time)	.371	1.06	24	15	.47

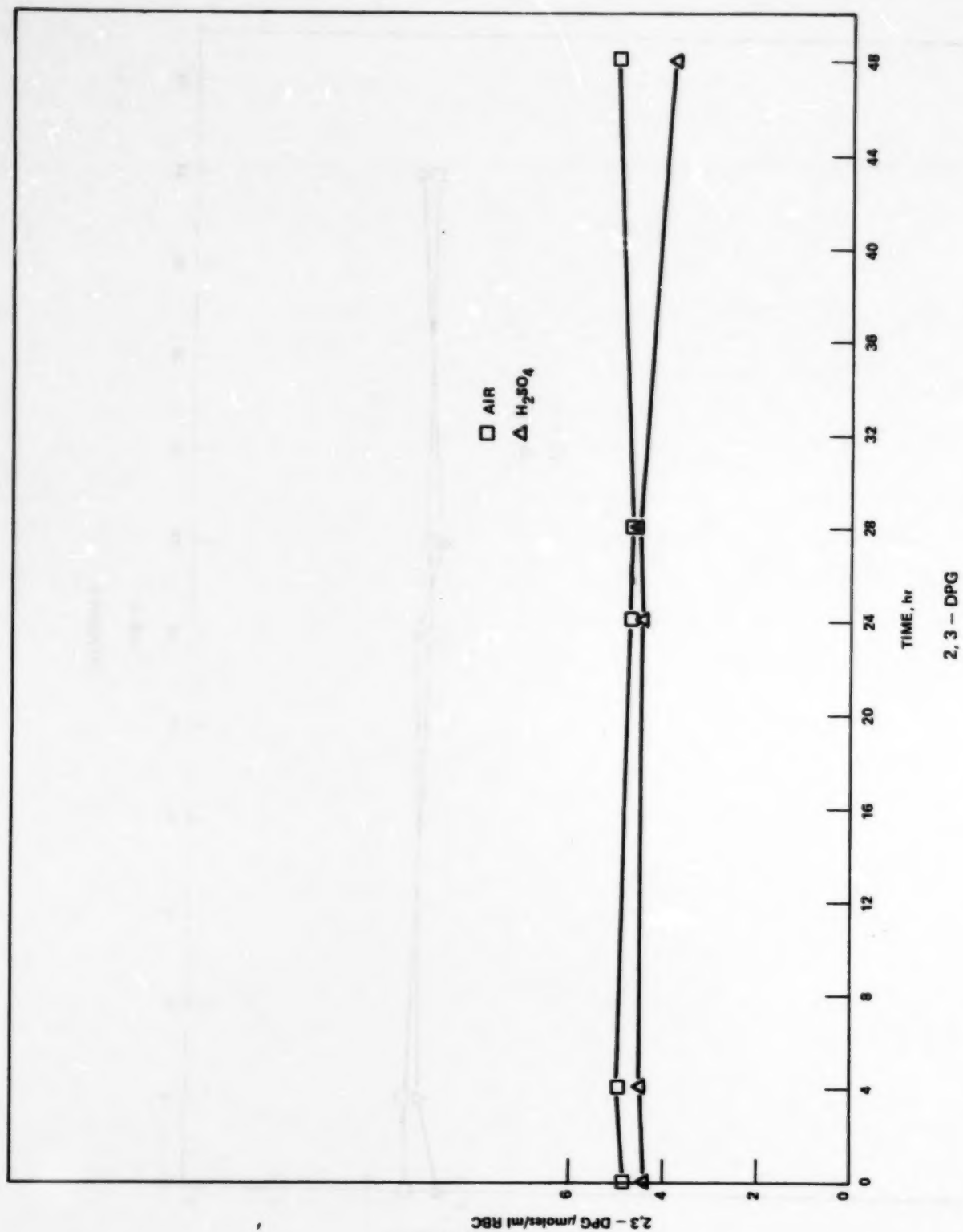
*Rao's F approximation

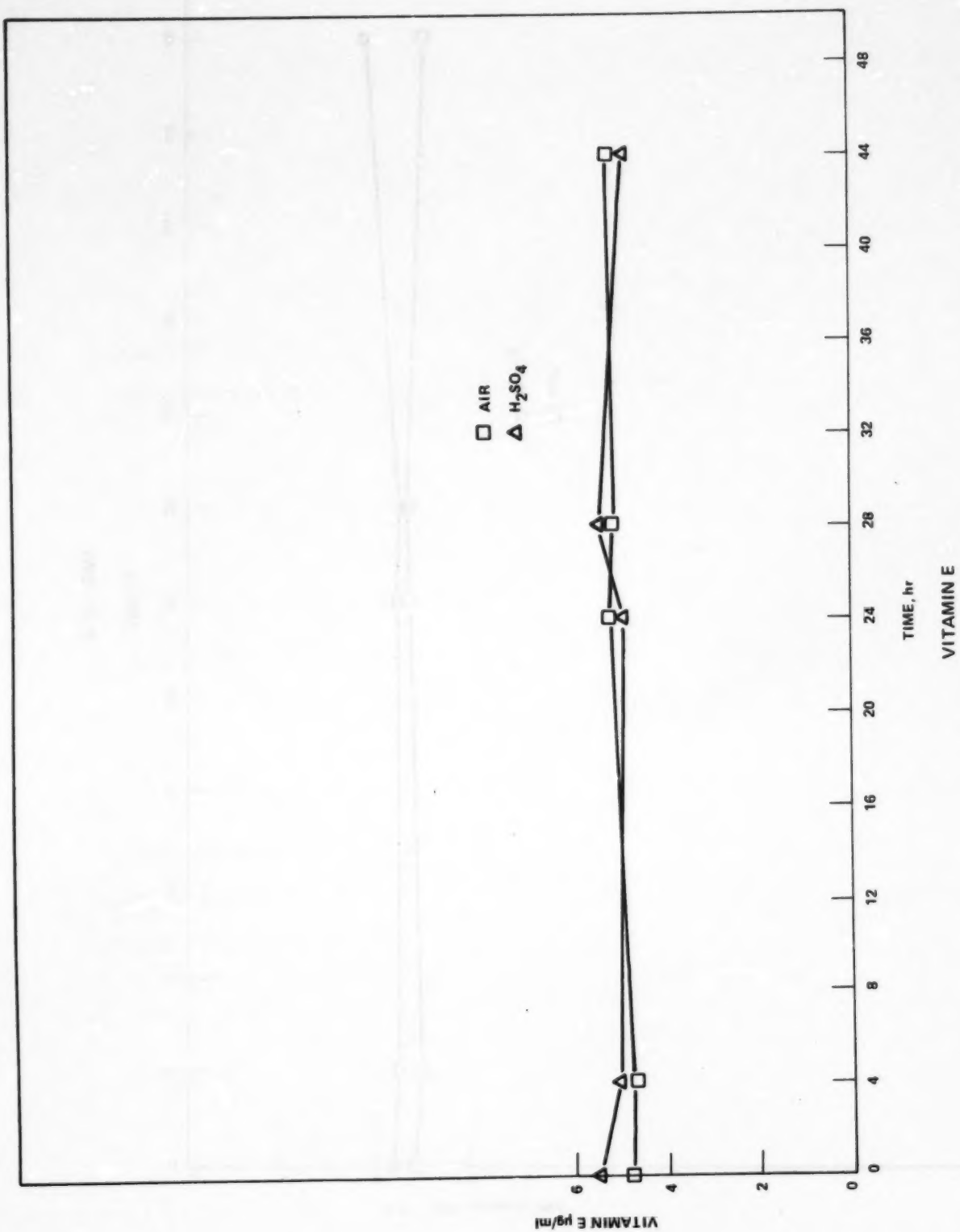
TABLE 3
Mean Response on All Seven Dependent Variables
Estimated from MANOVA

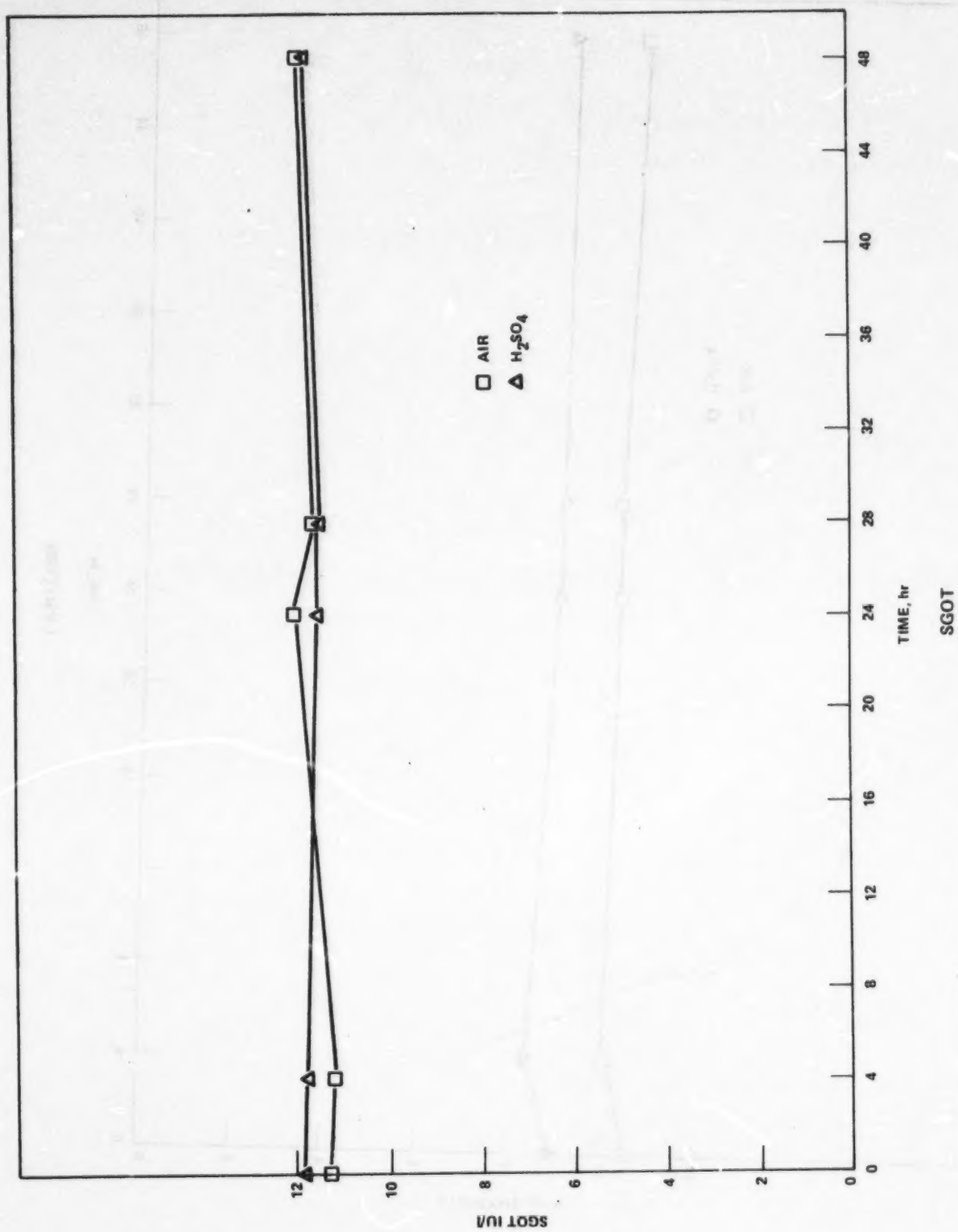
Variable	Treatment	Day 1		Day 2		Day 3
		Pre	Post	Pre	Post	Pre
Glutathione mg/100 ml	Air-air	38.60	39.82	40.25	40.68	39.03
	Acid-acid	38.32	39.52	39.41	42.00	38.61
Glutathione Reductase μ mole/gm Hgb/min	Air-air	8.22	8.15	8.41	7.82	7.83
	Acid-acid	6.80	7.33	7.08	7.41	7.16
RBC G6PDH U/gm Hgb	Air-air	2.06	2.05	2.08	2.09	2.20
	Acid-acid	2.03	2.01	2.01	2.09	2.15
Lysozyme μ g/ml	Air-air	10.56	10.09	10.28	10.26	10.68
	Acid-acid	8.91	8.35	8.96	9.01	9.03
SGOT IU/l	Air-air	11.3	11.2	12.0	11.5	11.8
	Acid-acid	11.9	11.8	11.5	11.4	11.7
Vitamin E μ g/ml	Air-air	4.89	4.84	5.13	5.15	5.12
	Acid-acid	5.49	5.03	4.97	5.34	4.87
2,3-DPG μ mole/ml RBC	Air-air	4.86	4.97	4.75	4.68	5.02
	Acid-acid	4.40	4.50	4.49	4.61	3.86

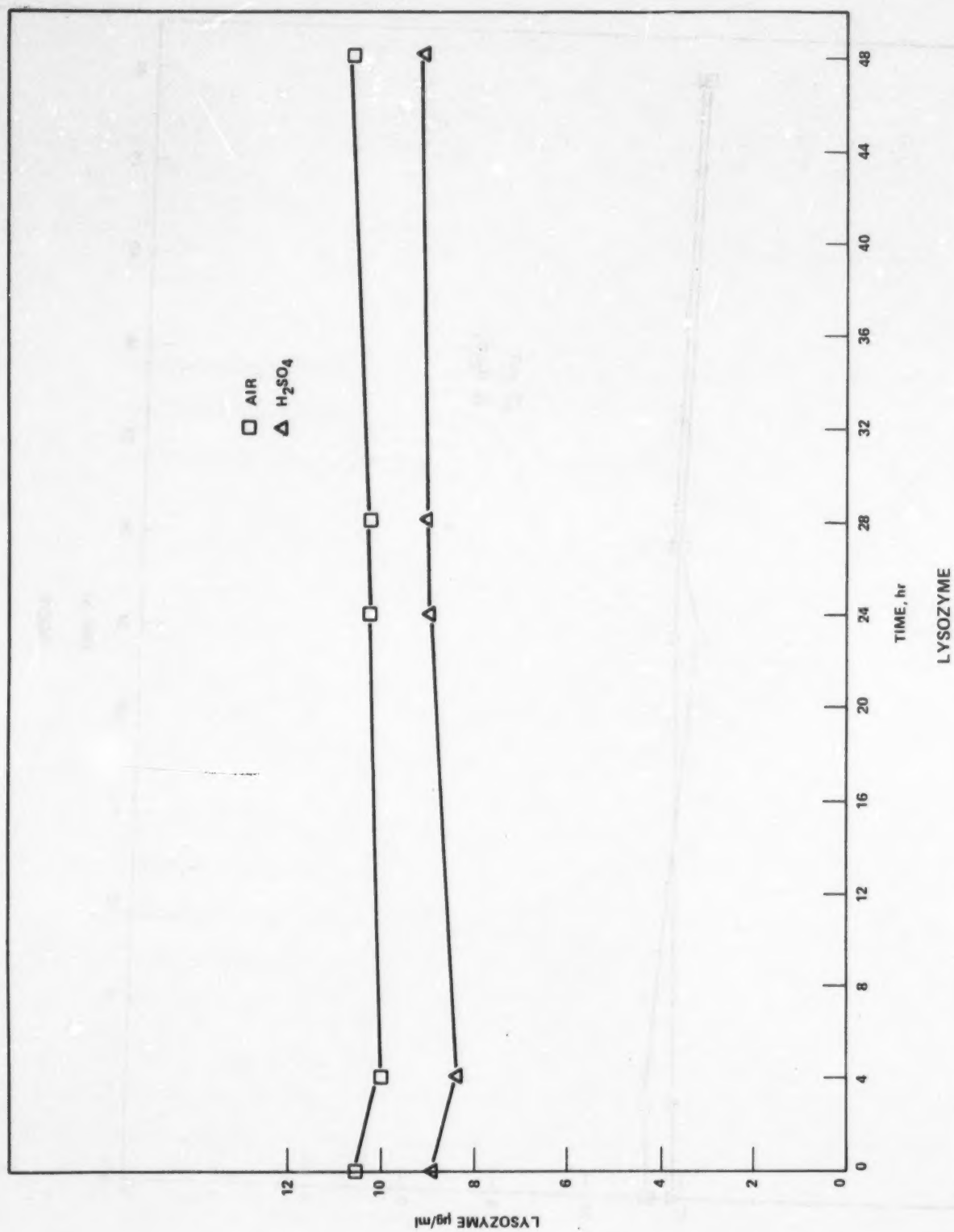
Figures 1-7: Acid by time means for: 2,3-DPG, Serum Vitamin E, SGOT, Lysozyme, RBC G6PDH, GSH, and RBC GSH Reductase.

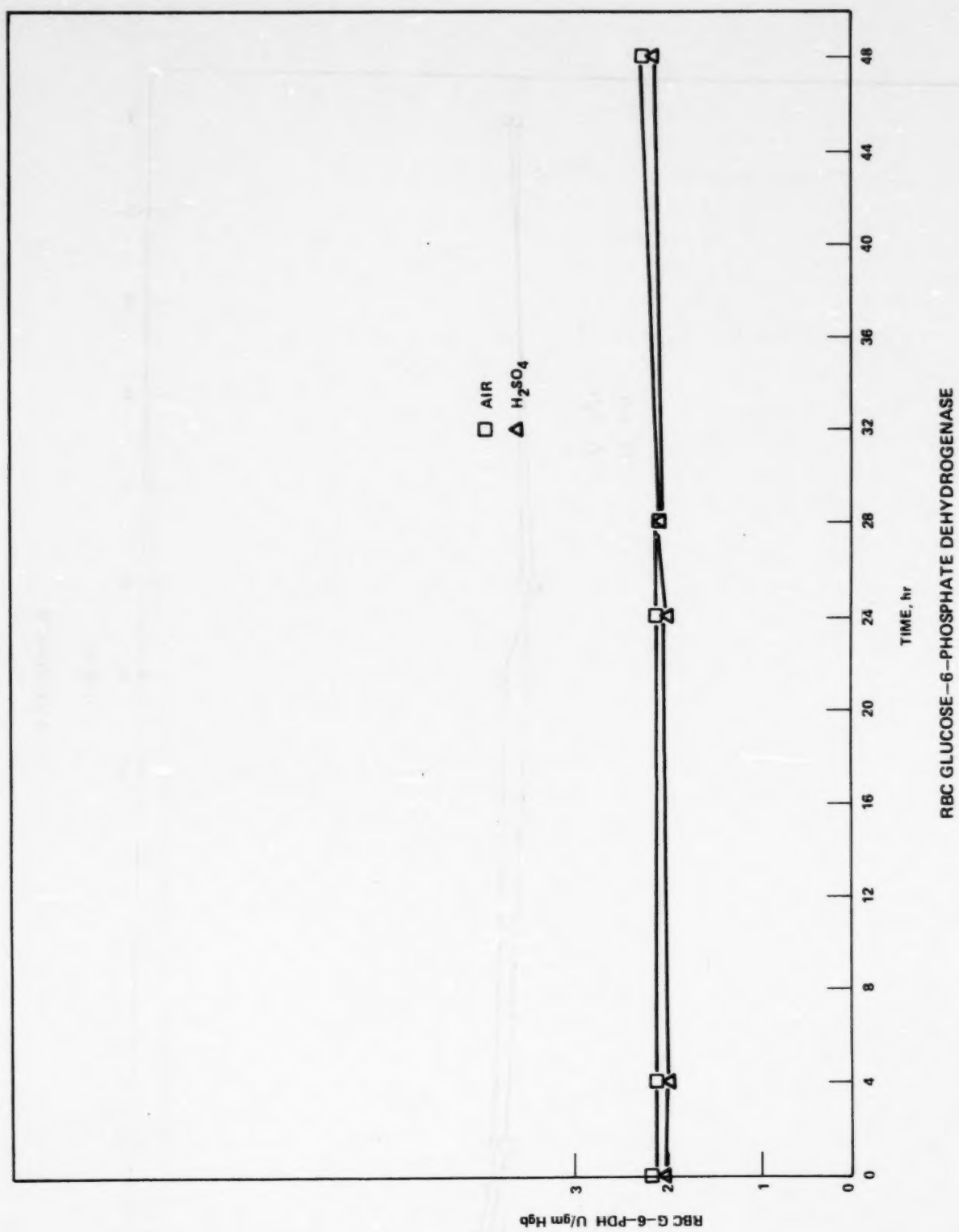
Time	2,3-DPG	Serum Vitamin E	SGOT	Lysozyme	RBC G6PDH	GSH	RBC GSH Reductase
0	18.75	10.25	27.25	1.47	15.25	1.47	1.47
15	18.50	10.25	27.25	1.47	15.25	1.47	1.47
30	18.75	10.25	27.25	1.47	15.25	1.47	1.47
45	18.75	10.25	27.25	1.47	15.25	1.47	1.47
60	18.75	10.25	27.25	1.47	15.25	1.47	1.47
75	18.75	10.25	27.25	1.47	15.25	1.47	1.47
90	18.75	10.25	27.25	1.47	15.25	1.47	1.47
105	18.75	10.25	27.25	1.47	15.25	1.47	1.47
120	18.75	10.25	27.25	1.47	15.25	1.47	1.47
135	18.75	10.25	27.25	1.47	15.25	1.47	1.47
150	18.75	10.25	27.25	1.47	15.25	1.47	1.47
165	18.75	10.25	27.25	1.47	15.25	1.47	1.47
180	18.75	10.25	27.25	1.47	15.25	1.47	1.47
195	18.75	10.25	27.25	1.47	15.25	1.47	1.47
210	18.75	10.25	27.25	1.47	15.25	1.47	1.47
225	18.75	10.25	27.25	1.47	15.25	1.47	1.47
240	18.75	10.25	27.25	1.47	15.25	1.47	1.47
255	18.75	10.25	27.25	1.47	15.25	1.47	1.47
270	18.75	10.25	27.25	1.47	15.25	1.47	1.47
285	18.75	10.25	27.25	1.47	15.25	1.47	1.47
300	18.75	10.25	27.25	1.47	15.25	1.47	1.47

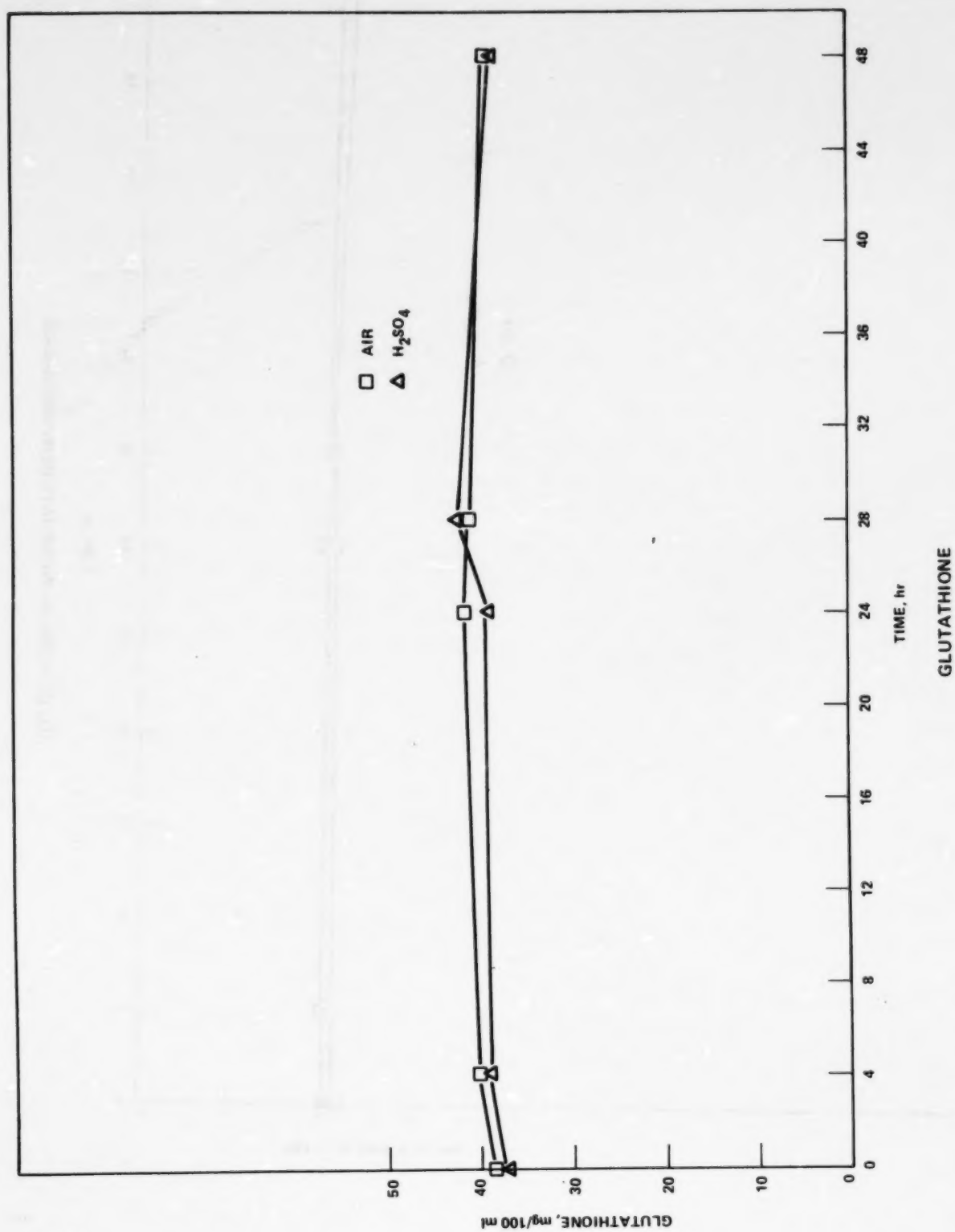


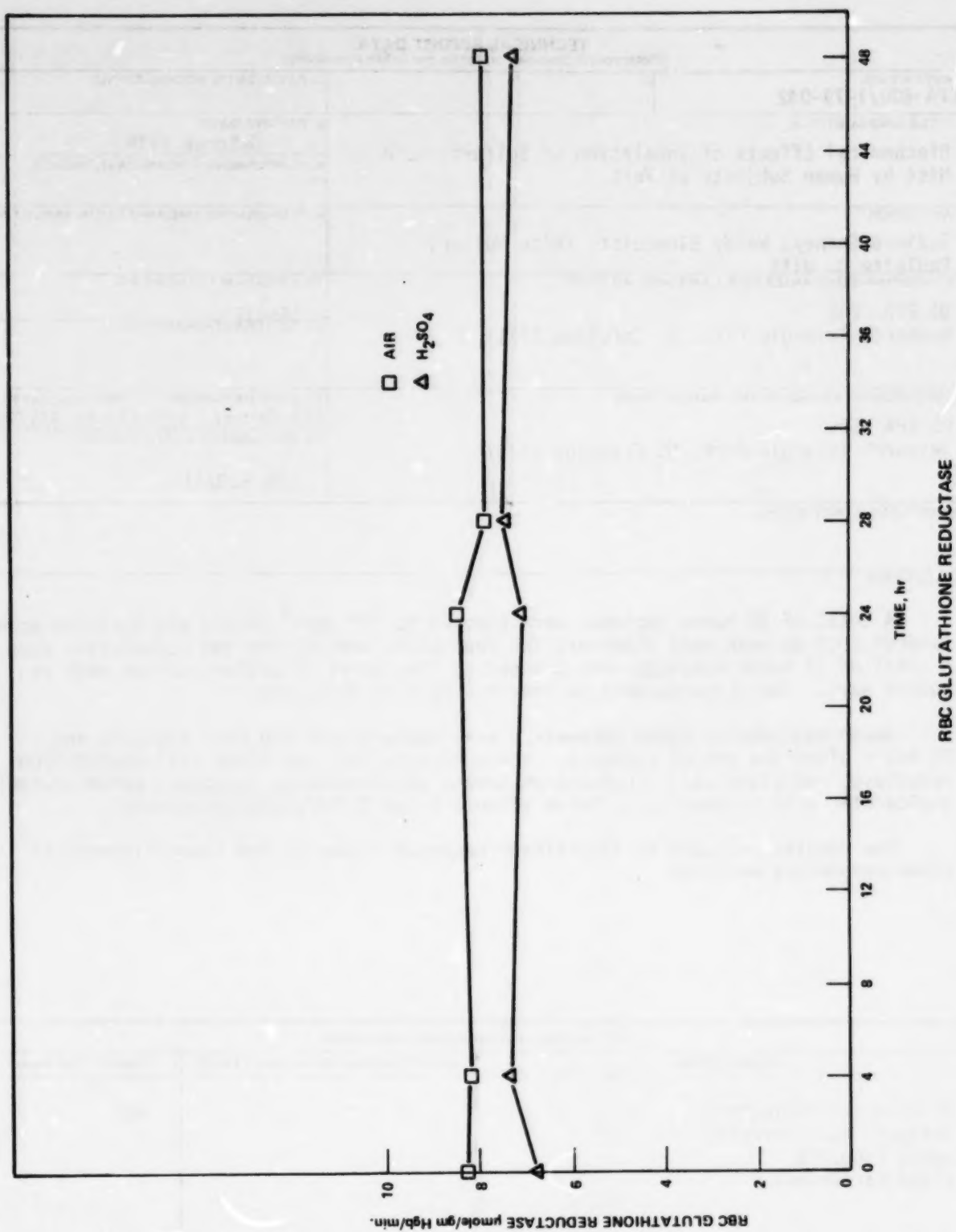












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